Amendments to the Specification:

Please replace the paragraphs beginning at page 23, lines 1-12 with the following amended paragraphs:

Figure 2 shows the PCR primers used for genomic PCR-SSCP of SCN1A (SEQ ID NOs: 99-188);

Figure 3 shows the sequence of the SCN1A mutations found in epilepsy patients (SEQ ID NOs: 189-192 and 309);

Figure 4 shows the PCR primers used for genomic PCR-SSCP of SCN2A (SEQ ID NOs: 193-306);

Figure 5 shows the mutation found in epilepsy patients in SCN2A (SEQ ID NOs: 307 and 308);

Figure 6 shows the PCR primers used for genomic PCR-SSCP of SCN3A (SEQ ID NOs: 310-399); and

Figure 7 shows the mutation found in epilepsy patients in SCN3A (SEQ ID NOs: 400-408).

Please replace the paragraph beginning at page 52, line 3, with the following amended paragraph:

Genomic DNA form IGE and normal patients was obtained by conventional methods. Primers used to amplify the genomic DNA are shown in Figure 2. Following PCR, SSCP analysis was performed and mutations in SCN1A were identified as follows (Figure 3):

(1) Glu1238Asp; normal: GCA TTT GAA GAT ATA; (SEQ ID NO: 189) patient R10191 who has an idiopathic generalized epilepsy (IGE): GCA TTT GAC GAT ATA (SEQ ID NO: 190) found in 1 of 70 IGE patients). The mutation is thus a conservative aa change, in the extracellular domain between III-S1 and III-S2. Furthermore, this residue is located at the junction between the TM domain and the extracellular domain. It may thus influence gating

activity. The aa change between adult and neonatal isoforms is at a similar juxta-TM domain position (between I-S3 and I-S4).

(2) Ser1773Tyr; normal: ATC ATA TcC TTC CTG (SEQ ID NO: 191), patient R9049 (affected with IGE): ATC ATA TmC TTC CTG :(TCC>TAC,. (SEQ ID NO: 192) This mutation is in the middle of IV-S6 TM domain; found in 1/70 IGE patients, and 0/150 control subjects tested. This mutation is interesting from a biological point of view for a number of reasons. First, this region of SCN gene (IV-S6) has been found to play a critical role in fast inactivation of the SCN, by mutagenesis experiments in rat SCN (McPhee et al., 1998). This is highly relevant for pathophysiology of epilepsy, since this may increase neuronal hyperexcitability. Moreover, in patients with GEFs, a mutation has been found in the SCNB1 subunit, causing impairment of the fast inactivation of the SCN (Wallace et al, 1999). Finally, many of the antiepileptic drugs (e.g. phenytoin, carbamazepine) primarily act by reducing the repetitive firing of neuron, which also involves fast inactivation of the SCN.

Please replace the paragraph beginning at page 53, line 3, with the following amended paragraph:

Genomic DNA form IGE and normal patients was obtained by conventional methods. Primers used to amplify the genomic DNA are shown in Figure 4. Following PCR, SSCP analysis was performed and mutations in SCN2A were identified as follows (Figure 5):

(1) Lys908Arg: normal: TAC AAA GAA (SEQ ID NO: 307) for patient numbers always preceded by R; R9782 (Patient with IGE): TAC AGA GAA (SEQ ID NO: 308). The mutation is thus a conservative aa change, located in an extracellular domain between TM domains IIS5 and IIS6; in 1/70 IGE patients; 0/96 normal controls. The mutation involves an important component of the SCN gene, since the S5 and S6 segments are thought to form the wall of the transmembrane pore which allows the sodium to enter the cell. This may have an influence on the gating control of the pore.

(2) Leu768Val, in individuals R8197, R9062 and R9822 (all IGE patients) (found in 3/70 IGE patients and 0/65 control subjects). The mutations is in the IV-S6 component of the sodium channel, which is important in the inactivation of the channel (see above for more detail).

Please replace the paragraph beginning at page 53, line 21 to page 54, line 15, with the following amended paragraph:

Genomic DNA from IGE and normal patients was obtained by conventional methods. Primers used to amplify the genomic DNA are shown in Figure 6. Following PCR, SSCP analysis was performed and mutations in SCN3A were identified as follows (Figure 7):

(1) Asn43DEL: allele 1: CAA GAT AAT GAT GAT GAG (SEQ ID NO:400); allele 2: CAA GAT --- GAT GAT GAG (SEQ ID NO: 401); in open reading frame deletes 1 aa: DNDDEN->QDDDEN, in the cytoplasmic N-terminal segment; in IGE patients, the frequency of allele 1 = 131/146 (0.90); allele 2= 15/146 (0.10); for IGE patients: homozygotes (22): R3958, R9632; heterozygotes (12): R9049, R9152, R9649, R9710, R9896, R10069, R10191, R10213, R9993, R10009, R10256. Of note, 2 patients are homozygous for the rare allele and all patients have IGE. In controls: allele 1 = 145/154 (0.94); allele 2 = 9/154 (0.06) and no 22 homozygotes were found.

(2) normal: tggtgtaaggtag (SEQ ID NO: 402), R10670 (IGE patient): tggtataaggtag (SEQ ID NO: 403), in conserved intron between 5N & 5A exons, significance uncertain.

(3) normal: ccccttatatctccaac (SEQ ID NO: 404), R10250 (IGE patient): ccccttatayctccaac (SEQ ID NO: 405); in conserved intron between 5N & 5A exons, significance uncertain.

(4) Val1035Ile: normal: AAA TAC GTA ATC GAT (SEQ ID NO: 406), R9269 (IGE patient): AAA TAC RTA ATC GAT, (GTA>ATA = Val>Ile) ;(SEQ ID NOs: 407 and 408). The mutation is thus a conservative aa change which destroys a SnaBI site (this could thus be used as a polymorphism identifiable by restriction enzyme digestion). In SCN1A, this Val is a Ile, therefore probably not a causative mutation. In cytoplasmic domain bw II-S6 & III-S1 TMs; found in 1/70 IGE alleles; and 0/70 controls.